Lecture summaries

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Heart-lung interactions

The heart and lungs are interconnected. The heart delivers blood to the lungs, and the lungs oxygenate blood and regulate its chemical composition. Mechanically, the lungs function as a negative-pressure (suction) pump, while the heart is a positive-pressure pump. Both reside within the thorax: intrathoracic pressures influence pulmonary and cardiac function, and cardiac and pulmonary pressures reciprocally affect each other – these are the foundations of heart-lung interactions.

During normal spontaneous breathing, negative intrathoracic pressure results in lung inflation *and* alters cardiac filling and emptying. Under positive pressure ventilation, the effects on filling and emptying are reversed.

This lecture will a) review the physiology of heart-lung interactions during spontaneous (negative pressure) *versus* positive pressure breaths, b) examine the clinical implications of heart-lung interactions in selected diseases, and c) explore how these principles may inform clinical decision making in critically ill patients.

Learning goals

1. Define heart-lung interactions and explain their mechanisms.
2. Explain the cardiopulmonary differences between spontaneous (negative pressure) and positive pressure breaths.
3. Understand the role of heart-lung interactions in selected diseases.
4. Consider how we may use heart-lung interactions to guide clinical decision-making.

Extra-pulmonary effects of mechanical ventilation

Positive pressure ventilation (PPV) can be life-saving and effective use requires detailed understanding of pulmonary physiology. However, a lung-centric approach overlooks the extra-pulmonary consequences of PPV. In particular, the cardiovascular, renal and neurologic systems are fundamentally – and often adversely – affected by PPV. Appreciating these effects is essential to managing the whole patient, not just the lungs.

In this lecture we will review how PPV affects the cardiovascular, renal and neurological systems, and consider how we might use this knowledge to better manage our ventilated patients.

Learning goals

1. Describe the effects of positive pressure ventilation on the cardiovascular, renal, and neurologic systems.
2. Consider ways to mitigate adverse extra-pulmonary effects during positive pressure ventilation.

Non-cardiogenic pulmonary oedema: pathophysiology and treatment

Non-cardiogenic pulmonary oedema (NCPE) is the accumulation of interstitial and/or alveolar fluid not primarily due to elevated capillary hydrostatic pressure, as in congestive heart failure. The principal mechanism is increased endothelial permeability, though other factors may contribute. While elevated left atrial pressure may coexist – particularly with iatrogenic fluid overload or concurrent cardiac disease – it is not the initiator of oedema in NCPE, unlike in cardiogenic pulmonary oedema (CPE).

In NCPE, protein-rich fluid extravasates into the interstitium and alveoli. Normally, interstitial fluid is cleared *via* the lymphatics and alveolar fluid clearance depends largely on ion transport, especially *via* basolateral Na+-K+-ATPase activity in pneumocytes.

As a blanket term, NCPE encompasses all oedema not caused by CPE; multiple mechanisms and aetiologies apply. This lecture will review endothelial barrier physiology, contrast the pathophysiology of NCPE and CPE, and examine specific NCPE syndromes and treatment options.

Learning goals

1. Describe normal endothelial fluid physiology.
2. Compare the genesis of non-cardiogenic *versus* cardiogenic pulmonary oedema.
3. Review specific scenarios leading to non-cardiogenic pulmonary oedema.
4. Consider treatment options for non-cardiogenic pulmonary oedema.